TABES AND MUSCULAR ATROPHY.[1]

By Joseph Collins, M. D., New York.

PROFESSOR OF NERVOUS AND MENTAL DISEASES IN THE POST-GRADUATE MEDICAL SCHOOL; VISITING PHYSICIAN TO THE CITY HOSPITAL; CONSULTING NEUROLOGIST TO THE HOSPITAL FOR RUPTURED AND CRIPPLED.

Some degree of muscular atrophy is not uncommon in tabes, in fact such complication occurs in from 15 to 20 per cent of all the cases. It is responsible for many of the deformities of the feet which occur late in tabes, such as the tabetic clubfoot and sometimes for the total motor incapacity amounting to paralysis. The variety or form of atrophy that I shall consider particularly in this article, however, is not the slighter forms that lead to deformity of the feet and hands. I shall consider rather the muscular atrophy that is occasionally encountered with tabes, which resembles in its distribution and course progressive muscular atrophy of spinal origin. The other forms of progressive muscular atrophy have recently been described by me in an article in the *Medical News* dealing with the Symptomatology of Tabes. The variety of atrophy of which the cases that I publish herewith are examples has nothing peculiarly characteristic of it in its clinical manifestations, so far as I can make out, save that it overshadows the tabes, which is rarely typical, in its clinical delineation. The atrophy may attack any part of the body, and extend so as to involve all of the extremities, as it did in my first case, or it may confine itself entirely to the upper extremities, as it did in my second case, or it may be localized, as in my third case. Occasionally, amyotrophy in tabes, which is probably not unlike in its causation the form that I am describing, is limited to a smaller area than in the third case, for instance, the half-sided atrophy of the tongue that was described by Charcot as a complication of tabes may be of the same origin, and likewise the atrophy of the soft palate that has been mentioned by Marie and Koch.

When the extensive amyotrophies of tabes were first described by Condoleon, Leyden, Charcot and Pierret, it was generally accepted that they were dependent upon alteration of the anterior horn cells, either parenchymatous decay or diminution in number. Some of the cases reported by these authors, as well as those of Chretien and Thomas, prove that tabetic amyotrophy is in some instances dependent upon cytological changes in the anterior horn cells. On the other hand, during the past fifteen years a very considerable number of cases have been reported in which disease of the anterior cornual cells has not been found. The muscular atrophy in these cases is adequately explained by the lesions of the peripheral nerves that accompany them. So today, we must admit that amyotrophy of a very profound type occurs with tabes, and that it is dependent in some instances upon lesions of the anterior horn cells, in others, the more frequent, upon disease of the peripheral nerves. It is true that in some cases of extensive muscular atrophy in tabes we have, as Dejerine has suggested, a coincidence of tabes with chronic poliomyelitis, just as occasionally we have it with other chronic nervous disease, such as paralysis agitans (Placzek), and multiple sclerosis (E. W. Taylor). I see no reason why a patient with tabes should not develop paralysis agitans, or multiple sclerosis, but of course, there is no relationship between them. Slight alteration of the peripheral nerves is not infrequently found when the lesions of tabes are studied with the microscope[2] and a theory of the pathogenesis of tabes has been founded in part upon the existence of these lesions. The changes in the peripheral nerves in tabetic amyotrophy are of a different nature from those found in tabes with slight muscular atrophy, and the lesions of the former cannot be looked upon as an intensification of the process in the latter. As a matter of fact, the slight alterations of the peripheral nerves that have been described in some cases of tabes may occur in almost any slowly progressive central nervous disease of protracted duration. In the severer forms, such as Case I in this series, the lesion is a true parenchymatous inflammation or degeneration. Some writers have endeavored to make the changes in the peripheral nerves secondary to functional affection of the anterior horn cells, for instance, Schaffer[3] maintains that the anterior horns are functionally damaged consecutive to degeneration of the collaterals of the posterior roots. The cellular affection begins with very delicate structural changes which develop slowly. The clinical character of tabetic atrophy is in complete harmony with the cellular changes. The most characteristic feature is its slow progression, the absence of fibrillary twitchings and of reaction of degeneration. The most common location is in the peroneal region. Schaffer records a case in which Argyll-Robertson pupil, loss of the patellar reflexes on the right side, and a tabetic arthropathy of the left hand were the symptoms. The peroneal muscles were paralyzed on the left side. Histological examination showed that the motor cells of the lumbar cord were very much diseased, while those of the cervical cord were normal. These changes were made out by the use of Nissl's method. The author looks upon the peripheral nerve lesions as secondary or dependent upon the central. Such an explanation may suffice for the minor tabetic amyotrophies, but it is entirely inadequate for the varieties considered here.

---

[1] Read at the meeting of the American Neurological Association.

---

[2] See article on "Morbid Anatomy and Pathogenesis of Tabes," Medical News, April, 1903.

[3] Revue Neurologique, 1896, No. 4.

The history of the first patient whose case I shall describe in detail is as follows:

Mr. M. was first seen by me December 28, 1895; he was then forty-seven years of age. His personal history previous to the beginning of his present illness was that he had married when twenty-five years of age and had been a widower since he was forty. He denied having had syphilis, but stated that all his life he had taken the risk of such infection. He admitted having had gonorrhea. In his business, that of a merchant, he had been a fairly hard worker, and during the last four or five years he had been worried because of business reverses. He was temperate in the use of spirits and an abstainer from tobacco. His family history was that his father and mother had died of apoplexy, the former at the age of sixty-five, the latter at the age of seventy-six. They had three children, one of whom died in infancy and the other in late childhood from acute diseases. So far as the patient knows there is no history of nervous disorder in the family. His own marriage had been childless.

Mr. M. enjoyed good health until about his forty-third year, when he suffered from an attack of pleurisy and pneumonia, and although he does not attribute his present trouble to that disease, he says that he has never really been well since he had it. A short time before the pleuro-pneumonia he was thrown from a horse and fell on his back. So far as he knows this fall did not injure him in any way, as he got up and rode on. After the attack of pleuro-pneumonia he suffered a great deal with his throat and had to go frequently to a physician, but I am unable to find just what the nature of the trouble was. Two or three months after the attack of pneumonia he noticed for the first time that both hands felt somewhat numb, and that there was some loss of strength in the left hand. At first this was not heeded particularly, as the numbness was variable in its intensity. A few months later he noticed that spaces were beginning to form between the bones of the left hand, and after this atrophy of the interossei had become quite marked, weakness and atrophy of the muscles of the right hand developed. He says that when the shrinkage began to show itself in the right hand the left hand got somewhat better, but it is probable that it got better merely by contrast. Later the atrophy extended to the forearms, arms and shoulders. All this time, he says, there was no pain, merely the sensation of numbness mentioned above.

Coincident with the development of the trouble with the hands, and possibly even before it, the patient remarked that it had become impossible for him to urinate in the customary way. At first the stream could be started with great effort after waiting patiently for a considerable time, but soon it became possible only to urinate when at stool, and he soon fell into the habit of urinating twice a day, at which time he would take a copious irrigation of the rectum. He had been constipated for some time before the beginning of his present trouble. He had begun the injections only after the difficulty with urination showed itself, and since the same time he had been wholly impotent, occasionally having a semi-erection in the morning when the bladder was over distended. About this time he noticed a sense of insecurity in walking in the dark, and soon fell into the way of having his room lighted at night so that he would be able to walk about more securely when he had to get up. About eighteen months after the symptoms showed themselves in the hand the right leg became lame so that he couldn't raise the thigh, or cross the right leg over the left without manual assistance, and the leg dragged so that he had to lift the knee very much higher on this side than the other in order to swing the toe free from the ground; in other words, foot drop on this side was an early symptom. All this time there was very little pain. The extremity wasted from the hip to the toes. Six months later a similar condition in the left lower extremity; the lameness was not quite so marked, but the wasting was just as conspicuous. It was particularly striking below the knee. During this time the wasting and paralysis of the upper extremities progressed somewhat, but with nothing like the progress that they did in the lower extremities. The condition of the bowels and bladder remained about the same. There was no incontinency of urine. The patient was able to get about with the aid of two sticks.

The patient had been under treatment by general practitioners and specialists from the first manifestation of his trouble, but treatment had not been of service. He had received electrical baths, hypodermic injections of strychnine, pilocarpine, the animal extracts, electrical treatment and massage. Three months before coming under my observation he began to complain of pain in the lower part of the spine, and of a sensation as if swarms of ants were crawling beneath the skin in the feet and legs. It might here be added that this symptom became very severe and added greatly to the burdens of the patient's life. He thought that the wasting and the lameness had apparently come to something like a standstill, as he seemed to be able to get about nearly as well as he had been any time during the six months previous. He complained of failing sight, of occasional severe attacks of dizziness, of involuntary twitching of the legs and drawing up of the knees. After being in bed a few hours the legs and thighs suddenly and forcibly flex, then extend, then the toes draw up and the feet twist about. These involuntary movements often occur every fifteen minutes, beginning at about 2 A. M. until he arises. They deprived him enormously of strength.

He does not complain of fibrillary twitching either when he is lying down or when he is quiet. The legs feel unwieldy and the feet very uncertain in their action. The only complaint of the upper extremities is that he cannot use the right hand very well. His business at this time compelled him to use a pen nearly all day, and the hand cramped so severely after a half hour or so that he would have to wait until the muscles got rested. Despite this he thinks that the hands are better now than they were six months ago. The left upper extremity is fairly strong and it does not give him any trouble.

On examination it is seen that the lower extremities and muscles about the pelvis, lower abdomen and right hand are the seat of marked wasting or atrophy. The remainder of the body seems fairly well nourished. The atrophy is most marked in the right hand, which has lost its normal contour, the thenar and hypothenar eminences being entirely flattened and the interosseous spaces hollowed. He is unable to adduct the fingers after they have been abducted, and to reflex the first phalanx of the thumb on the first metacarpal bone. The terminal phalanx of the thumb tends to remain in a position of forced extension. There is similar wasting, but to a much less degree in the left hand. The relative strength of the two hands is shown by the dynamometric registrations, that of the right hand being 20, that of the left hand 50. Although the upper extremities are thin, there is no real atrophy of any muscle or group of muscles. The direct myotatic irritability of the muscles of the upper extremities is lively, but the contractions produced by the percussion hammer are slow and vermiform. The wasting in the lower extremities is most advanced in the muscles supplied by the sciatic nerves, the right leg is affected more than the left. When he attempts to stand with the feet together the ankles tend to turn out and the knees to bend, and unless he is very careful he falls. The atrophy has begun to show itself in the shape of a pelvic girdle, i. e., extending around the upper part of the thighs and the lower part of the trunk. He experiences a great deal of difficulty in flexing and extending the thighs, but the adduction and abduction of the thighs is still performed very well. The most difficult movement of the feet for him to make is extension, especially of the right foot, which has assumed the position of foot-drop. On attempting to walk without shoes the right foot wobbles uncontrollably and is very apt to overthrow him. The electrical irritability of the muscles is characteristic of partial reaction of degeneration, i. e., there is less irritability to the faradic current than to the galvanic current; although both of them produce a response, that to the faradic is extremely sluggish and vermiform. The pupils are typical Argyll-Robertson, they are small, almost pin-point and wholly immobile to light and shadow, they contract in accommodation. There is no ataxia of the upper extremities. The sensory reflexes, plantar, cremasteric and abdominal are present and lively, save the lower abdominal, which is rather sluggish. Sensibility of the lower extremities is not acute, but still it was impossible to say that there was any real anesthesia or analgesia at this time. There was tenderness on deep-seated pressure over the sciatic and popliteal nerves.

The patient was put under treatment, which consisted of lenient massage, faradism to the muscles, galvanism to the spine, hypodermic injections of strychnine, and otherwise a general tonic plan of treatment. He grew worse gradually and notes of a complete examination made eight months afterward, viz., on the 1st of September, 1896, show that the patient walks laboriously with the aid of two sticks. The portions of the body in which the atrophy has made the most striking progress is in the lower pelvic girdle. The gluteus maximus muscles especially seem to have very nearly disappeared; there is very little power in this portion of the body. The movements of walking, swinging the legs, etc., are accomplished very largely by the aid of other muscles. The atrophy in the pelvic girdle is not quite so conspicuous on the anterior surface as it is on the posterior surface. The legs below the knees are in sharp contrast with the upper part of the thigh, insomuch as the atrophy here is relatively slight. The toes of the right foot are in a more or less constant state of flexor cramp, and he has very little voluntary power to move or wiggle them. Electrical examination of the abductors and extensors of the thighs shows that there is no response to the faradic current, while the anterior and internal crural muscles show a sluggish and vermiform response. The reaction to the galvanic current is diminished.

The immediate muscular irritability of the lower extremity is very sluggish, and no tendon response can be made out. The patient complains of paresthesia in the feet and along the outside of the thigh, and there is distinct analgesia over the peroneal nerves. Involuntary drawing up of the legs at night is quite as troublesome as it was.

The bowels and bladder do not act save on direct stimulation. It is necessary to inject large quantities of water into the rectum and colon before an evacuation can be secured. The bladder empties itself in part during defecation, but it is now necessary to use the catheter regularly. The atrophy of the muscles of the hand and right upper extremity has apparently ceased to progress. About this time the patient began to have disagreeable sensations of paresthesia and pain in the lower lumbar and sacral region.

The patient's condition grew gradually, very slowly but steadily worse. When he first came he was able to walk with a stick, a year later he was able barely to get about with two sticks, and needed great assistance in getting in and out of a car or carriage. A year later he could drag himself around upon crutches, and in another year he was in a wheel-chair. A note made April 12, 1898, states that the pain in his back is so severe that he cannot bear the weight of a truss which he has had to wear because of a right inguinal hernia. The left leg is now quite as much involved as the right. The toe-drop and foot-drop are so complete in each extremity that he stumbles and falls over everything. The bones of the lower extremities feel, he says, as if they had been beaten with heavy weights. The muscular twitching in both legs and in the right hand is extremely distressing, the fingers are much weaker, but the atrophy does not seem to have made any progress, it still confines itself to the thenar and hypothenar muscles of the first interosseous space. The formication on the bottom of the feet and the peroneal nerves, particularly the right, is intolerable. Vision has failed very much during the past year; now everything seems to be cloudy and misty. Examination of the optic nerve shows distinct optic atrophy, the pupils are the same as before, the bladder now can be emptied only by catheterization. The patient until this time had been of good courage, and faced his increasing infirmities bravely; he now became wofully despondent in the face of physical impotency and financial distress. He lasted about a year longer, during which time there was no particular change in his symptoms, the atrophy did not become more advanced than is indicated above, but he seemed to grow gradually weaker. A few months before his death he had some paroxysms of difficulty in breathing, but they were evidently symptomatic in character. The patient died rather suddenly, the symptoms immediately preceding death being those of pulmonary edema.

The spinal cord, pieces from many of the affected muscles and sections of the musculospiral, ulnar, sciatic and popliteal nerves were removed and prepared for microscopical examination.* The spinal cord was most profoundly affected in the lumbar region, and the morbid process diminished in intensity and extent upward from this region. Examination of sections taken from the first lumbar segment shows a degeneration of the posterior columns, and to a less extent of the posterior part of the lateral columns and the posterior roots. The bulk of the degeneration is borne by the middle third of the columns of Burdach and by the posterior roots. The lesion, which is a degeneration of the axis cylinders with a comparatively slight increase of glia tissue and very little vascular degeneration, is not, however, confined to that part of the column of Burdach to which the name "bandelettes externes" is given by the French writers. This part of the column is degenerated, but so also, particularly higher up, is the posterior part of the column of Burdach and the column of Lissauer. There is in addition some degeneration of the lateral part of the cord, especially of the posterior part of the cross-pyramidal tracts. The process does not seem to have involved all of the pyramidal tracts, nor is the intensity of the degeneration so great here as in the posterior columns. The involvement of this part of the cord is most evident in the upper lumbar region, least evident in the dorsal region, and slight in the cervical region. The uncrossed pyramidal tracts are not affected. There is no degeneration of other white columns of the cord. The anterior gray matter of the spinal cord in the lumbar region shows no abnormality, it seems to have preserved its contour and its normal constitution. The cell bodies of the peripheral motor neurones are of the customary size and shape and number, and accept the Nissl stain in the customary way. There are no vascular changes in the gray matter worthy of note. Many of the cells of the posterior columns are degenerated, atrophied and shrunken. The peripheral part of the posterior gray substance shows a greater amount of degeneration than the central part. There is little or no evidence of disease of the meninges.

Sections from the dorsal region show a different picture from that just described. The degeneration here is confined almost exclusively to the columns of Goll. The picture that is presented is almost identical with that of ascending degeneration in this column produced experimentally by section of posterior roots in the lumbar region. This degeneration of the columns of Goll can be traced upward into the cervical region, diminishing in extent, however, as we go upward, i. e., the degeneration occupies relatively a less area of these columns. In the lower part of the dorsal cord the columns of Burdach show slight degeneration, especially in that part of the column which borders on the posterior horn; in other words, the zone of the entering posterior roots. But as I have said, this area of degeneration soon disappears. The other parts of the cord are fairly intact. The lateral columns do not seem to be quite as normal in their appearance as they might be, but it is impossible to say that there is any degeneration of the axis cylinders here. The anterior gray matter is normal in its appearance and constitution, the columns of Clarke are well preserved, and the posterior gray matter presents no detectible change.

---

*Reference to the mode of preparation is omitted. All the modern methods of staining the nervous tissues were used.

The state of the cord in the cervical region needs no extensive description. There is no lesion to be made out save the small area of degeneration in the columns of Goll corresponding to the degeneration spoken of below.

Examination of the spinal ganglia of the dorsal and lumbar region, stained with hematoxylin and, according to Nissl's method, did not reveal degeneration of the cell constituents of the ganglia, despite the fact that the degeneration of the posterior root fibers in the supra-ganglionary area was very evident. The anterior gray matter of this part of the cord was studied most carefully, especially sections stained by Nissl's method, but no alterations that are admitted to be truly pathological were found. Some of the cells showed a fair degree of chromatolysis, others irregular pigmentation, and here and there slight vacuolation. Dr. B. Onuf, who kindly looked through the specimens, subscribes to the statement that these cells could not be considered to be the seat of pathological alteration.

Sections of the ulnar, the median, the popliteal and other nerves show them to be the seat of alteration of the nature of parenchymatous and interstitial degeneration. The larger part of the nerve is transformed into fat tissue and fibrous interstitial tissue. Many of the nerve fibers that are not so transformed show partial degeneration. The fibers are swollen, twisted, here and there beset with fat globules. The walls of the blood vessels are thickened, particularly the external and middle coats, and the lumen is somewhat decreased. These thickened blood vessels are in striking contrast with the fat masses into which so much of the nerve has been transformed. The intensity and character of these alterations and changes in the nerve are more satisfactorily conveyed by the accompanying illustrations than by verbal description. In some of the nerves the process was more advanced than in others; for instance, in the ulnar and external popliteal it was most advanced. In the former more than one half of all the fibers are transformed into a homogenous fat mass, while sections from the sciatic nerve show that fibers having a more or less normal appearance predominate numerically, although the degeneration is here very striking. The sensory nerves seem to be involved only to a very slight degree. Sections from the radial and peroneal nerves show that the diameter of the nerve fibers is variable, and that the interstitial tissue is somewhat increased, but there is little or no alteration in the axis cylinders or in the sheaths of Schwann. The degeneration of the nerves is to be seen distinctly in many of the muscles. For instance, in the lumbricales. Here the irregular aspect and ragged appearance of the nerve fibers are very apparent, likewise the scarcity of them.

The lumbricales muscles fibers are themselves the seat of parenchymatous and interstitial alteration, but the process is neither so well marked nor so extensive as in the nerve. The muscle spindles are normal. This is well shown by Plate D, which shows two muscle spindles with thickened capsules. Between them a degenerated small nerve bundle cut obliquely is to be seen. Sections from the muscles of the thenar eminence, and from the gluteal muscles show most satisfactorily the change that went on in the muscles. It consists of a degeneration and atrophy of the muscle fibers, an increase of the interfascicular fibrous tissue, and a thickening of the blood vessels, but without proliferating endarteritis or anything suggestive of an obliterating process in the blood vessels. There is a very considerable fatty infiltration of the fibers, more in the sections from the gluteal muscles than from those of the hand.

The case therefore is one of tabes with muscular atrophy of neural origin, the lesions of the peripheral nerves being a degenerative neuritis. The lesions of the muscles are very similar to those of neuropathic atrophy, a parenchymatous decay with accompanying fatty infiltration and slight connective tissue increase. The clinical features of the tabes were not typical, neither were the lesions of the cord. In my study of the profounder forms of tabetic amyotrophies, I have been very much struck by a statement made by Chretien and Thomas[4] to the effect that there is a form of tabes that begins with muscular atrophy in which tabes symptoms remain very much in the background. In none of the three cases that I am reporting in this article was the existence of tabes suspected until it was revealed by the physical examination. Strümpell, in commenting upon the cases of the above mentioned writers, remarks that paralyzed legs do not show ataxia. But that has nothing to do with the subject. The point is, that in these cases, such as I report, there are no subjective symptoms of tabes of any moment, and the disease tabes does not comport or display

---

[4] Revue de Médecine, 1898, p. 886.

itself in the ordinary way. The best proof of this is that tabes is only revealed by the examination. It may be that the atrophy is such a dominant symptom that it obscures the tabetic symptoms. But the atrophy does not always come first in these cases. For instance, in a case somewhat similar to mine reported by Goldscheider,[5] the following symptoms developed first: Girdle-feeling, diplopia, right-sided ptosis, numbness in the legs. At the time of examination there were present: Right-sided ptosis and mydriasis, ataxia of the lower extremities with profound muscular atrophy, slight ataxia of the hand, sensory disturbances in the lower extremities, and loss of knee-jerks. In the course of the disease the atrophy became more and more pronounced, and the picture of tabes quite typical. The patient was a woman forty years old who denied syphilis and alcoholism. On microscopical examination there was found in the middle of the cervical region degeneration of Goll's column, and likewise a similar area which extended into the anterior portion of Burdach's column, reaching the lateral border between the middle and posterior third of this. This area was separated from Goll's column and from the posterior horn by an area equally wide of intact white matter. The degeneration increased as the examination was carried lower and lower, so that in the dorsal region nearly all of the posterior columns was involved by the degenerative process. About six cm. above the beginning of the lumbar cord there was a limited degeneration of the lateral columns especially at the periphery. This area was larger in the lumbar region and two cm. below the beginning of the lumbar segments it had the appearance of a wedge driven into the lateral part of the cord. In a number of peripheral and sensory motor nerves there was found intense atrophic degeneration. Other cases of tabetic amyotrophy in which more or less involvement of the lateral part of the cord has been found, are reported by Schultze and Strümpell.

In this JOURNAL of February, 1894, I reported a remarkable case of progressive muscular atrophy with locomotor ataxia, which it may be said in passing has been referred to by some European writers as one of probable syringomyelia. If one is willing to make the diagnosis of the latter disease when the symptoms of tabes and progressive muscular atrophy co-exist, I cannot gainsay him the privilege. I had the case under observation for many years and saw him last three years after the report of the case was published. During this time no features diagnostic of syringomyelia were present.

---

[5] Zeitschrift f. klin. Med., Vol. xix.

The patient, a man fifty years old, had been a hard drinker in his early life, and had contracted syphilis when twenty-five years old. When he was thirty-six years old, and at a time when he was drinking freely of alcoholic liquors, he noticed for the first time that the arms and the legs were getting weak, and that he had occasional attacks of pain in the legs. Two years later he lost control of the bladder, and was so weak that he had to go to a hospital. While he was in the hospital his upper extremities became quite powerless, the hands and shoulders began to get smaller, and continued to do so until they reached the condition in which I found them, absolute powerlessness and flaccidity of the upper extremities. Soon after he went into the hospital he suffered from incontinence of feces as well as of urine. After two years in the hospital, he had regained control of the sphincters, and his legs became stronger. It was at this time that he began to complain that he saw double, and of lightning pains in the lower extremities. These pains were so severe that for weeks at a time he could not sleep. Sexual power was lost rapidly, and he had occasional incontinence of urine. From this time he was unable to walk in the dark and had difficulty in descending steps. He says he feared that he would fall as he could not grasp the bannister with the hands. The wasting of the upper extremities, which had been progressing during this time, began first on the right side, and has always been more marked there, although the left is extremely affected. The wasting involved the flexors and extensors equally and no suspicion of the "claw hand" ever developed. The wasting at first was rapid, but after three to four years it progressed very slowly.

Examination made in April, 1893, showed: On inspection, the patient when at his ease assumes a posture with the head bent forward so that the chin nearly touches the chest. (Photographs of the patient are to be seen in the original article.) When asked to hold the head erect the chin is tilted upward and the head is inclined a little backward. The former is his habitual posture. The upper extremities hang like flails from the shoulders. They are completely powerless. By swaying the body he is able to start them in a swinging motion. The hands look as if they had been flattened with a rolling pin; the eminences and depressions have entirely disappeared. The forearms and arms have lost almost completely their muscular tissue, some fat still remains. The head of the humerus on the right side has dropped from its socket so that a depression the width of a finger is left. On the left this is also apparent, but to a less degree. The muscles of the shoulders and back presenting the most extreme atrophy are, on the right side, the deltoid, supraspinatus, and infraspinatus, serratus anticus (which presents a remarkable degree of wasting), trapezius, with the exception of its occipital bundles, teres major and minor. The latissimus, pectoralis major and sternocleidomastoid are affected to a less degree. The rhomboids are quite well preserved when contrasted with the right.

There is no fibrillary tremor in any of the partially wasted muscles and no response to tapping. The size of the lower extremities is not far from normal, but there is a suspicion of atrophy in the left peroneal group.

There is a marked Romberg symptom, and inability to stand on one foot or to raise himself on tiptoes, and inability to walk backward. There is considerable ataxia of locomotion. The knee-jerks and ankle-jerks cannot be elicited. In the lower extremities there is marked diminution of sensibility, especially for pain, and sensation is greatly delayed. He says "now" in response to a severe prick of a pin in from three to four seconds in different parts of the lower extremities, and the maximum pain is not attained for several seconds. Sensibility of the upper extremities is well preserved. Temperature sensibility normal. The pupils react moderately to accommodation, but not to light. The patient complains of great heaviness of the lower extremities, paroxysmal lancinating pains, and of girdle sensation. Cold weather or exposure aggravates his symptoms markedly and causes frequently distressing weakness of the sphincters. The atrophy of the muscles has apparently come to a standstill.

The most legitimate interpretation to put upon this case, it seems to me, is that the patient had multiple neuritis probably of alcoholic origin, and that the motor nerves of the upper extremities and shoulders were so profoundly diseased that they never regenerated. Whereas, those of the lower extremities being very much less affected, quickly recovered. The most serious objection to this interpretation is that the sensory nerves of the upper extremity were spared. It may well be, however, that at the onset of the probable neuritic process they were affected as well. Despite the fact that the toxic effect of alcohol is most apt to be expended upon the sensory nerves, cases of alcoholic motor neuritis are not uncommon. If we do not adopt this idea of the muscular atrophy the case must be looked upon as one of muscular atrophy of spinal origin, and against this conception a number of factors speak. In the first place the suddenness of onset, rapidity with which it developed and destroyed the muscles, the uniform manner in which it affected all the muscles and its absolute cessation are very unlike the manner in which spinal progressive muscular atrophy comports itself. The most distinguishing clinical feature of the latter disease is the selective manner in which it destroys groups of muscles of an extremity, leaving others untouched. The insidious way in which it attacks the muscles, the existence of fibrillary twitchings, the course of the disease are all very different. The lesion may have been a chronic anterior poliomyelitis, in fact there is nearly as much to be said in favor of this diagnosis as of that of motor neuritis.

The third case of muscular atrophy associated with tabes that I shall speak of is one that is mentioned here more because of the interesting question in diagnosis that it presented rather than because it comes in the category of the cases that I have been describing: D. T., a barman, forty-two years old, contracted syphilis at the age of twenty-four, and had very little specific treatment. A year later he had an attack of iritis. When thirty-four years old he had a cerebral seizure in which he was very dizzy and fell to the ground but was not unconscious. For two days he did not speak correctly. There was no paralysis accompanying or following the attack. Before this attack he had suffered for a number of months with headache. Four years later he had a similar attack. His present illness was preceded for a number of weeks by mental depression. On August 18, 1902, while eating supper, he fell from his chair to the floor. He was only partly unconscious he says, for he knew those that helped him to bed. The following day the right arm and leg were equally paralyzed and he had dysarthria, but no real aphasia. When he came to the hospital the hemiplegia had so far recovered that he was able to walk slowly with the aid of a stick. The degree of recovery was greater in the leg than in the arm. On examination of the right upper extremity a marked wasting of the muscles of the shoulder cap was seen. This was so great that the head of the humerus dropped from the glenoid cavity. There is no evidence of fibrillary contraction in the atrophied muscles, and they react sluggishly to the electrical currents, more so to the faradic than to the galvanic current. Further examination shows absent knee-jerks and ankle-jerks, pronounced hypotonia of the left lower and upper extremities, Romberg's sign, Argyll-Robertson pupils, diminished sensory acuteness over the circumference of the body, corresponding to the 9th and 12th ribs, and in the feet and legs. Plantar jerks exaggerated, both of the extensor type, the right constituting a typical Babinski phenomenon. The patient has scarcely any symptoms of tabes save impotency and occasional incontinence of urine, and the disease was not suspected until its existence was revealed by the examination.

The muscular atrophy which he shows may be explained in one of two ways, either it is dependent upon a peripheral lesion, such as a neuritis of the circumflex and other nerves of the brachial plexus, or it is due to a thrombosis of the cornual branch of the anterior spinal artery, similar to the thrombosis within his brain that caused the hemiplegia, with resulting limited neurone destruction. At the present time it is impossible to say upon what the atrophy is dependent. The fact that it seems to be slowly progressive speaks in favor of the latter view. The case is of interest in showing that an attack of cerebral apoplexy with consecutive degeneration of the crossed pyramidal tracts, as evidenced by the Babinski phenomenon, does not cause a return of the knee-jerk. In this case it is extremely probable that unequivocal signs of tabes antedated the attack of cerebral thrombosis upon which the hemiplegia is dependent.

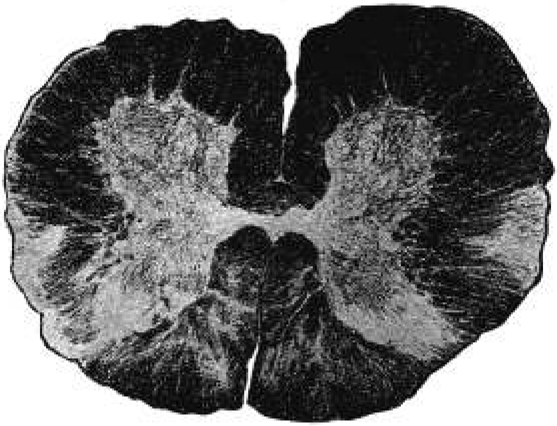


FIG. 1

Some of the cases of tabes and muscular atrophy that have recently been published in addition to those already mentioned, are the following:

Wagner[6] reports two cases. The first case, a thirty-six year old laborer, who had been ill eighteen months, said that weakness and wasting of the extensors of the right foot set in shortly after a course of mercurial inunctions. The affected muscles showed partial reaction of degeneration. The author thinks that the neuritis may have been of mercurial origin, but considering that the reality of mercurial neuritis is not generally granted, this seems very improbable.

---

[6] Inaug. Dissertation, Berlin, 1896.

The second case, a woman forty-seven years old, with signs of lues and symptoms of tabes, complained of weakness of both forearms; the thenar and hypothenar eminences were flattened, the interossei likewise atrophied, giving the typical claw-hand. There was partial reaction of degeneration in the nerves.

V. Ufen[7] reports a case of tabes dorsalis and muscular atrophy, in which there was wasting of the muscles of the hand, shoulder and the thighs. There were no electrical changes. At the anatomical examination there was found a well marked spinal meningitis in addition to the typical posterior cord root degeneration, but no atrophy of the anterior horns. The peripheral nerves were not examined, but Ufen does not doubt the neuritic nature of the muscular atrophy, as the anterior horns were normal.

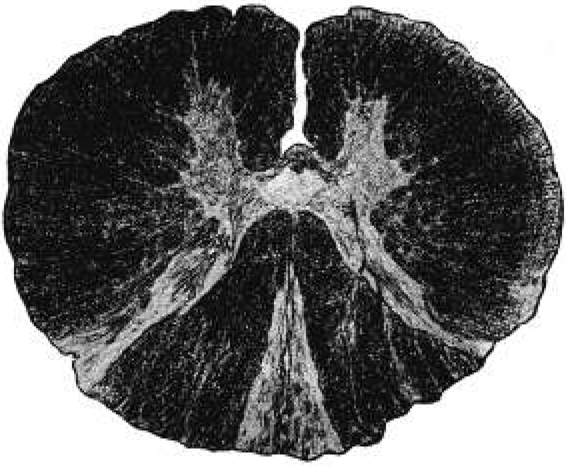


FIG. 2

Jolly[8] has recorded a case of tabes with muscular atrophy which was quite profound in one lower extremity and in both upper extremities. There was very little disturbance of sensibility in the arms.

---

[7] Inaug. Dissertation, 1896, Kiel.

[8] Berliner Gesellschaft f. Psychiatrie u. Nervenkrankheiten, March 9, 1891.

Chretien and Thomas[9]. Their first case in which there was atrophy of the lower extremities of rapid development and slight atrophy of the arms, showed on autopsy in addition to tabes, widespread degeneration of the ganglion cells of the anterior horns in the lumbar region. The changes in the peripheral nerves were distinct, but not so marked. In their second case the anterior horns showed only slight changes, whereas there was profound peripheral neuritis.

E. W. Taylor[10] has recently published a very striking case of progressive muscular atrophy of the muscles of the hands and forearms, which, from the clinical point of view, seemed to indicate degeneration of the peripheral motor neurones.

---

[9] Revue de Mèdecine, 1898, November, p. 886.

[10] Boston Medical and Surgical Journal, 1902.

EXPLANATION OF PLATES.

Plate A—Transection of the ulnar nerve showing the greater part of the nerve transformed into fat tissue and interstitial fibrous tissue. Also the enormously thickened blood vessels, take up a considerable portion of the tissue that remains. Of the still remaining nerve bundles many show considerable degeneration.

Plate B—Showing four degenerated nerve bundles, A, B, C, D, in a lumbricalis muscle.

Stained after Pal counterstained with Picro-acid-Fuchsine.

The nerve fibers show accordingly as black stripes in the longitudinally cut bundles B, C, D. Note the ragged appearance and scarcity of nerve fibers in the four degenerated bundles A, B, C, D, especially in A and B.

Fat=fat tissue.

Plate C—From the same section as Fig. B. showing, also in a lumbricalis muscle, numerous degenerated nerve bundles in which the scarcity and irregular ragged aspect of the nerve fibers are very evident.

Verm.=vermiform muscular fibers.

Fat=fat tissue.

Atr. M.=atrophic muscular fibers.

M. transv.=transversely cut muscular tissue.

Plate D—Taken from a lumbricalis muscle from the same section as plate B and C. It shows two muscle spindles, with thickened capsula, but apparently well preserved muscle fibers. Between the two muscle spindles lies a degenerated little nerve bundle, cut obliquely.

M and M are two muscle fibers of the lumbricalis muscle.

PLATE A.

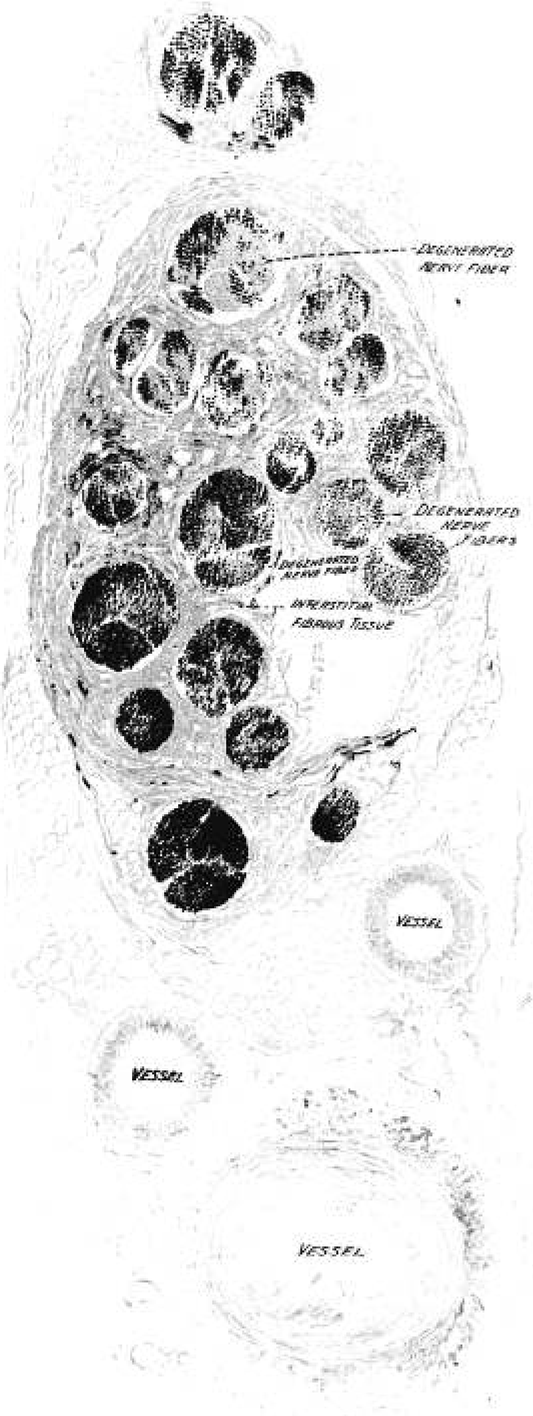
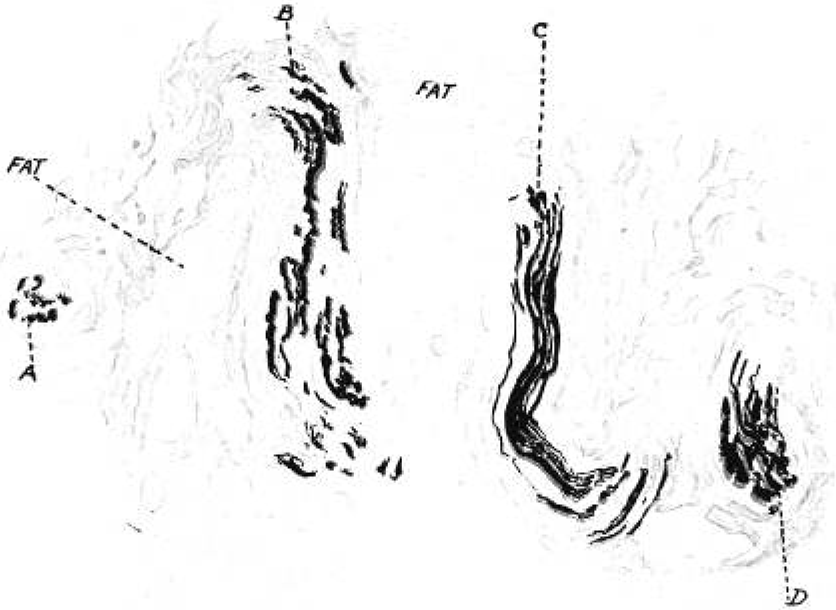
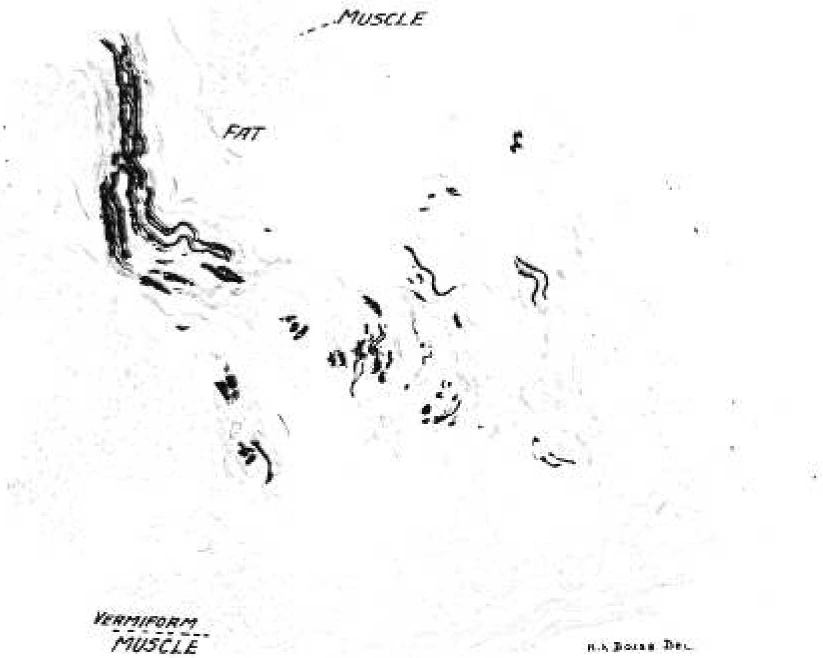

DEGENERATED NERVE FIBER
DEGENERATED NERVE FIBERS
DEGENERATED NERVE FIBER
INTERSTITIAL FIBROUS TISSUE
VESSEL
VESSEL
VESSEL

Plate B.

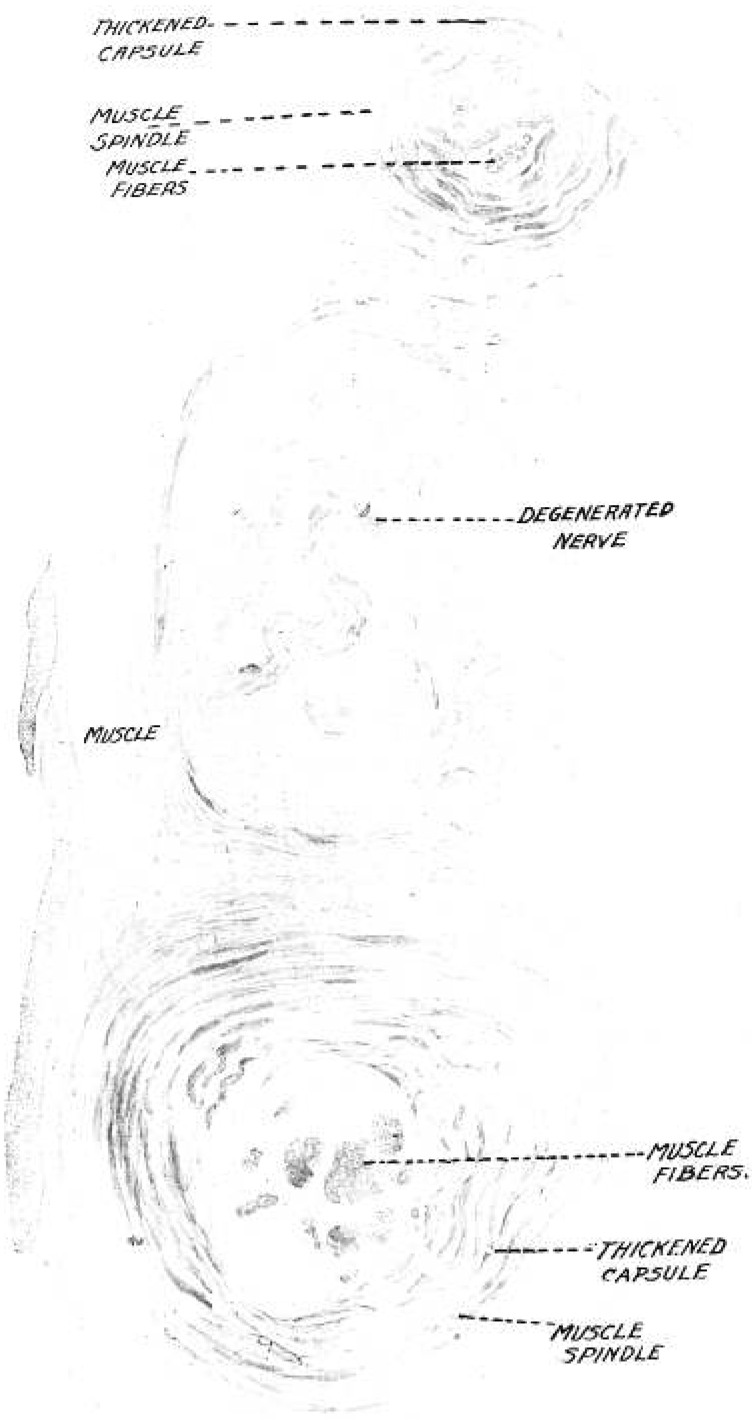




H. A. Boise Del.

Plate C.

Plate D.


THICKENED CAPSULE
MUSCLE SPINDLE
MUSCLE FIBERS
DEGENERATED NERVE
MUSCLE
MUSCLE FIBERS.
THICKENED CAPSULE
MUSCLE SPINDLE